is a pure infection by the time it has reached its fastigium is not a part of the present discussion, but it is a question which must be considered in drawing deductions from the treatment of typhoid fever with vaccines.)

Typical infections of the second class are no more difficult to treat than those of the third class. The diagnosis is made and the vaccine injected—as soon as the ferments have been produced in sufficient quantity the infection is overcome and the patient recovers. Such simple technique is effective in relatively few diseases—those in which vaccines are at present considered indispensable. But we must not conclude that all other infections belong to the first class; by far the great majority, I think, should be placed in a subvariety of the second class. Although they may react to the mere injection of vaccines with little or no benefit, careful examination of other factors complicating the process will reveal the fact that with accessory methods of treatment, positive results may be obtained.

An interesting illustration of a complicating factor is seen in pyorrhea alveolaris; vaccine treatment alone has little local effect, but when foreign bodies, such as calculi, are removed and the pus pockets carefully cleansed, the results are highly satisfactory. In this case, the complicating factor has been recognized and methods for its successful removal have been elaborated. There are, however, other infections which have not received the careful study devoted to pyorrhea alveolaris. If we are to expect further progress in vaccine therapy, this progress must come chiefly, as I have striven to indicate, through investigation of accessory methods in treatment.[2]

KERATOSIS PILARIS OF THE SCALP.

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In 1869 Hermann Beigel[1] described a peculiar disease of the scalp which was characterized by the presence of small, whitish, nit-like masses on the shafts of the hairs. Although Beigel's description was written nearly half a century ago, it is so accurate and concise that it may profitably be quoted in full: "The hair appears perfectly intact and normal in texture, but imbedded in a white, transparent, apparently structureless formation of a very peculiar nature. The knots are large enough to be seen by the naked eye, some measuring the tenth of a line in length. They adhere by no means firmly to the hair, but are rather loosely attached to it, and may be stripped off easily. Under the microscope their formation in regular layers, something like the slates of a roof, is very striking. Although some of the divisions are a little narrower than others, yet the whole formation gives the impression of great regularity. There appears to be, as already mentioned, no particular structure, the stratum being only finely dotted and here and there shriveled. This is when the low power is used. But on applying the higher power to one of these knots, prepared for microscopic examination by the application of water or glycerin, and exercising some pressure on the cover-glass in order to produce a thin layer, a structure of a well-known kind becomes visible. The whole knot, thus prepared, is found to consist of cells identical in structure with those of the inner transparent root sheath. . . . There remains no other explanation but one, that a hyperplastic action—perhaps consequent upon irritation or inflammation—exists in the sheaths of the roots of the hair, producing an abnormal number of cells, which are glued together and adhere to the cuticle of the hair while passing through the hair sac."

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[2] Since the presentation of the above paper the discovery of amebæ in pyorrhea has been announced by Allen J. Smith and M. J. Barrett. This is apparently opening up a new field in etiology, as the work done so far seems to indicate that amebæ constitute a determining cause in pyorrhea and possibly other infections. In its bearing upon the views presented in the above paper this observation is of more than passing interest. In amebic pyorrhea it is entirely possible that the bacteria found in the pockets may be largely enclosed within the amebæ. After digestion of the bacterial proteins within the amebæ the toxic portion alone is thrown off as such, the other fractions being still further degraded in the metabolism of the amebæ before their elimination. If such a condition should exist it would explain one of the factors complicating successful treatment of the disease by bacterial vaccines and call for the accessory use of an amebicide—like emetin.

[1] The Human Hair, Henry Renshaw, publisher, London, 1869.

Grindon[2] described a typical example of the disorder in 1897, and emphasized the differences existing between it and trichorhexis nodosa, monilethrix, the tinea nodosa of Cheadle and Morris,[3] Giovannini's[4] disease, leptothrix, piedra, and the chignon disease of Beigel,[5] which is due to the so-called "chignon" fungus (an organism probably related to, if not identical with, the one found in piedra). In Grindon's case the attack was not preceded or accompanied by seborrheic dermatitis, and there was no history or signs of alopecia pityroides. Grindon suggested the name "ecbolic folliculitis" for the condition, from the fact that the mass of cells was thrust out of the mouth of the follicle.

Recently I had an opportunity to study two cases of this disease, occurring in a mother and her four-year-old daughter. Neither had ever had prolonged arsenical or other treatment. The symptoms were practically identical in the two instances, and one description will suffice for both. The woman was a brunette, a housewife, aged twenty-five years; referred to me by Dr. Howard L. Snyder, of Winfield, Kansas. For several months she had been troubled by the occurrence of small, white, bead-like masses on the shafts of the hairs of her scalp. The adherent bodies gave rise to no subjective symptoms, but they bore a striking, if superficial, resemblance to the ova of pediculi, and caused the patient much annoyance. A thorough general examination revealed nothing of importance except a slight dryness of the skin. On the external surfaces of both the thighs and the upper arms were considerable numbers of the small, hard, conical papules which are characteristic of keratosis pilaris. The dorsal aspect of the fingers was normal, and there was no suggestion, either here or elsewhere, of pityriasis rubra pilaris or of xeroderma. The patient's hair was long and silky, and there were no signs of seborrheic dermatitis of the scalp. At several points on the scalp there were small, circumscribed, nutmeg grater-like keratotic areas. The hypercornification was confined to the mouths of the hair follicles, and gave rise to no itching or other disagreeable sensation. There was no alopecia as in Grindon's case. Strung along at irregular intervals on many of the hairs were hard, oval masses of inspissated epithetial cells. So closely compressed were many of the shafts that when the corneous material was forcibly removed the hair was broken off short at the lower margin of the encompassing mass. A number of the affected hairs, together with one diseased follicle and its accompanying horny plug, were secured for laboratory study. Nothing new was discovered concerning the cocoon-like collections of cells on the shafts. Numerous culture experiments with the material proved practically negative, only a few abbreviated chains of streptococci being obtained from one specimen. The piece of tissue was hardened in formalin, mounted in paraffin, and sectioned

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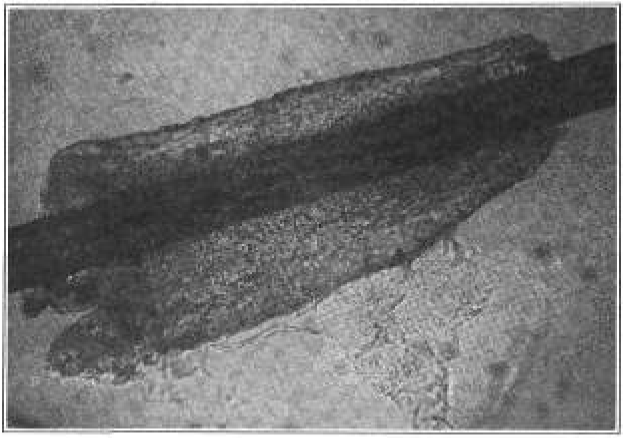
[2] Jour. Cutan. Dis., 1897, p. 256.

[3] Lancet, 1879. i, 190.

[4] Arch. f. Derm. u. Syph., 1887, xiv, 1049.

[5] Loc. cit.



Keratosis pilaris of the scalp, showing epithelial mass on hair shaft (moderate magnification.)

serially. The papillary bodies in the vicinity of the follicle were hypertrophied and the interpapillary vessels somewhat increased in size, but there were no signs of the presence of acute inflammation. There was some thickening of the prickle layer, but its relations were unchanged and no inflammatory or degenerative changes were noted. The follicle itself was slightly atrophied at the base, and the hair shaft considerably shrunken. Unfortunately the horny plug at the orifice had become detached at some time during the preparatory process, and its exact relation to the main body of the specimen was more or less a matter of conjecture. The mouth of the follicle was dilated, the walls meeting the hair shaft at a wide V-shaped angle. The corneous layer, which still adhered to the hypertrophied epidermis, was thicker than that usually found in a normal appendage. The contiguous sebaceous glands were much smaller than usual, with apparently some condensation of the surrounding connective tissue. None of the sections were stained for bacteria. From a general view-point the picture was that of a keratosis pilaris lesion which had existed for some time, and in which either as a result of suprafollicular pressure or intrafollicular changes, atrophic changes had occurred.

TREATMENT. A stimulating antiseptic lotion containing mercuric chloride, chloral hydrate, spirits of formic acid, castor oil, and alcohol, to be applied once daily, at bedtime, and an ointment containing salicylic acid and ammoniated mercury in rose water ointment, to be rubbed into the scalp once each week, were prescribed. The patient was instructed to wash the scalp thoroughly with warm soft water and green soap at intervals of a fortnight. The use of this combination promptly relieved the symptoms, but sufficient time has not elapsed to enable one to judge the permancy of the result.

SERODIAGNOSIS OF RABIES: PRELIMINARY REPORT.[1]

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THE early diagnosis of rabies has been the object of investigation of many an experimenter. Pasteur relied upon rabbit inoculation for diagnosis, but this meant fifteen to twenty days until the patient knew whether to get treatment or not. In some cases, according to Schauder, rabbits inoculated with street virus may

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[1] Read before the Pathological Society of Philadelphia, May 14, 1914.